Available online at www.sciencedirect.com
ScienceDirect
Journal of Acute Medicine 6 (2016) 67–69
www.e-jacme.com

Case Report

Cardiac arrest during recovery after tilt-induced vasodepressor syncope in a 76-year old man

Dorota Zyśko [a,*], Artur Fedorowski [b,c], Richard Sutton [d]

[a] Department of Emergency Medicine, Wroclaw Medical University, Wroclaw, Poland
[b] Department of Clinical Sciences, Lund University, Clinical Research Center, Skåne University Hospital, Malmö, Sweden
[c] Department of Cardiology, Skåne University Hospital, Malmö, Sweden
[d] National Heart and Lung Institute, Imperial College, St Mary's Hospital Campus, London, UK

Received 11 August 2015; revised 3 April 2016; accepted 22 May 2016
Available online 25 August 2016

Abstract

A 76-year-old man with a history of frequent syncope in youth and in the last 5 years was referred for a head-up tilt test. During the head-up tilt test, he developed a classical vasovagal reflex syncope. On immediate return to the supine position, the patient regained consciousness. Shortly afterward, the heart rate slowed distinctly and the patient became unresponsive with undetectable blood pressure. Chest compressions were initiated with return of spontaneous circulation 2 minutes later. Thereafter, the patient regained consciousness without neurological sequelae. This case illustrates cardiac arrest due to prolonged vasovagal reflex.

Keywords: cardiac arrest; reflex syncope; resuscitation

1. Introduction

Long-term outcome of patients with asystole induced by tilt testing has been assessed to be good. However, the patient is in danger of circulatory collapse from prolonged bradycardia and hypotension in patients with severe vasovagal reflex.

2. Case Report

A 76-year-old otherwise healthy male, smoker, with a history of frequent syncope not occurring in clusters, provoked by emotional stress in youth, and ~15 transient losses of consciousness without obvious trigger in the last 5 years was referred for a head-up tilt test at a tertiary hospital. The patient was not taking any drugs. The syncope, often associated with traumatic injuries, adversely affected his quality of life and made him unwilling to leave home alone. The patient underwent an initial evaluation according to European Society of Cardiology guidelines,[1] including 24-hour Holter monitoring, echocardiography, and carotid Doppler ultrasonography, all of which were either normal or nondiagnostic for the etiology of syncope. The test was performed using the Italian protocol (nitroglycerin administered after 20 minutes of passive tilt) in a tilt laboratory with available resuscitation equipment.[1] The patient developed a mixed vasovagal reflex, Type 1, as defined by the Vasovagal Syncope International Study (VASIS) criteria.[1] He fainted with unmeasurable blood pressure, although the heart rate remained at 60 beats/min. On immediate return to the supine position, the patient regained consciousness and was able to answer questions, despite a systolic blood pressure of 79 mmHg. Shortly afterward, the heart rate slowed down, and neither pulse nor blood pressure could be obtained (Figure 1). The patient vomited and lost his consciousness again. Agonal breathing, undetected pulse and blood pressure, and incontinence of urine and stool were observed. Extracorporeal cardiac massage was performed.

* Corresponding author. Department of Emergency Medicine, Wroclaw Medical University, Borowska 213 Street, Wroclaw 50–556, Poland.
E-mail address: dzysko@wp.pl (D. Zyśko).

http://dx.doi.org/10.1016/j.jacme.2016.05.002

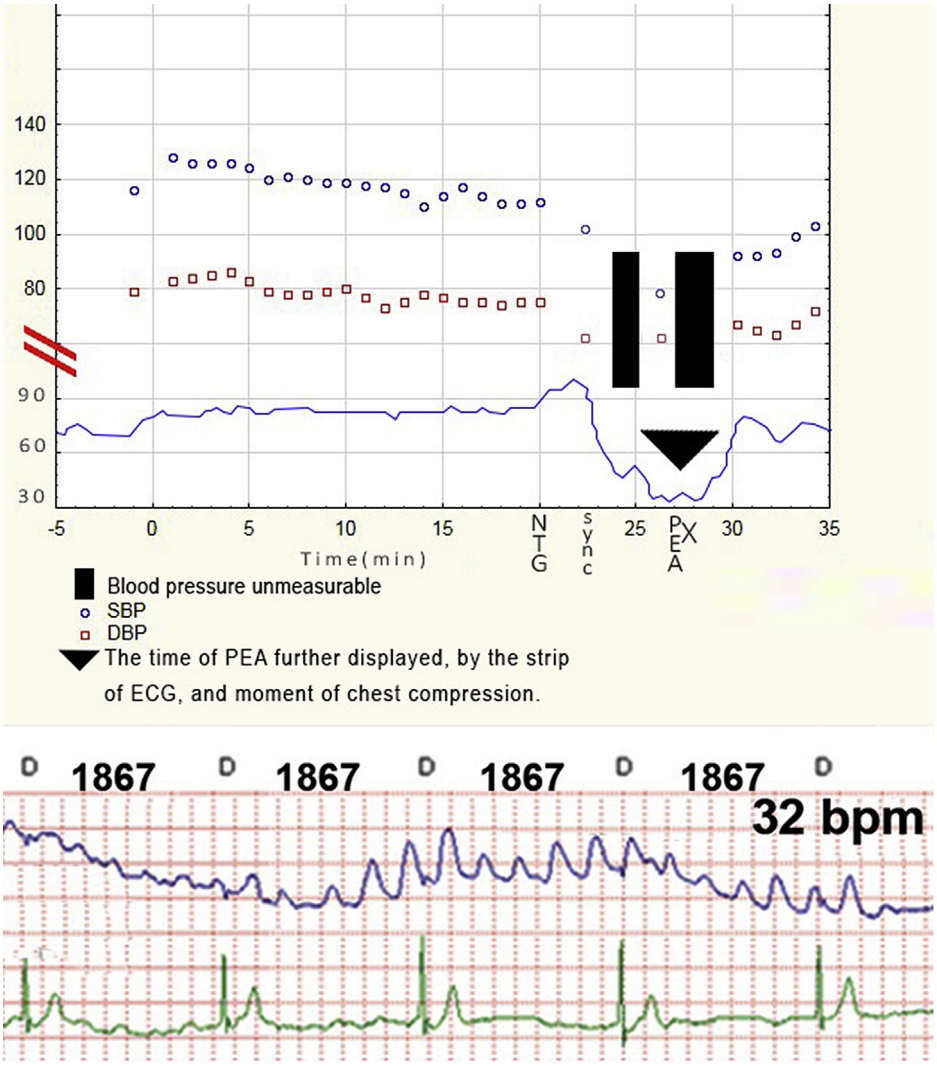

Figure 1. The patient's first syncope (VVS), which was tilt induced, and the second event in the recovery phase when in the supine position (PEA). The lower panel shows a three-lead ECG with chest compression during PEA (arrowhead). The longest sinus pause was 3.6 seconds. Bars indicate the periods without a measurable blood pressure. An arrowhead indicates the time of PEA, which is further displayed by the strip of ECG and moment of chest compressions. bpm = beats per minute; DBP = diastolic blood pressure; ECG = electrocardiogram; NTG = nitroglycerin; PEA = pulseless electrical activity; SBP = systolic blood pressure; VVS = vasovagal syncope.

Vein cannulation was installed, but intravenous atropine was unnecessary because the heart rate rapidly increased and spontaneous circulation resumed after 2 minutes; chest compressions were discontinued. Shortly thereafter, the patient regained consciousness and was subsequently referred for pacemaker implantation. During 24-month follow-up after pacemaker implantation, there has been no recurrence of syncope, although there has been one transient episode of severe dizziness with spontaneous recovery within 30 seconds. The patient has regained confidence and is now able to leave home alone.

3. Discussion

This case illustrates prolonged postsyncope hypotension described by Rozenberg et al[2] and Wieling et al[3] followed by sudden recurrence of syncope with severe cardioinhibition and cardiac arrest. The observed phenomenon is believed to be vagal in origin and is illustrative of a cluster of vasovagal attacks, as reported in some patients. After termination of the head-up tilt test-induced vasovagal reflex and resumption of the supine position, the heart rate and blood pressure usually return rapidly to baseline. Although postsyncope hypotension and/or bradycardia may last for several minutes in some cases,[3] the symptoms are usually mild and associated only with lightheadedness, nausea, and difficulty in maintaining an upright position. In this patient, the hemodynamic effects of sudden cardioinhibition and vasodilation led to a loss of consciousness and agonal respiration, which fulfilled the criteria for cardiac arrest. Agonal respiration is a sign of hypoperfusion of the brain center responsible for respiratory control and is a common finding at the onset of a sudden cardiac arrest.[4] It is also common in severe reflex syncope.[5] Return of efficient circulation was most probably related to

spontaneous resolution of the vasovagal reflex, including redistribution of blood volume by adoption of the supine position. However, it is difficult to determine what would have happened if the chest compressions had not been performed. During the tilt, the patient had received nitroglycerin, which may have reduced compensatory vessel responses compared with the spontaneous syncope reported by the patient. Atropine was considered as first-line therapy on the basis of the pathophysiology of bradycardia, occurring after vasovagal syncope. Atropine is recommended by current guidelines when cardiac arrest occurs in the operating room due to excessive vagal activity, and if this is the likely cause of the cardiac arrest, 0.5 mg atropine should be given intravenously and cardiopulmonary resuscitation should be started.[6]

According to recent publications, vasovagal reflexes in early life may predict both mortality and cardiovascular morbidity.[7–9] Moreover, frequent syncope independently increases the risk of cerebral white matter lesions.[10] This case may illustrate a mechanism by which "benign" vasovagal syncope may lead to major cardiovascular events including death in an age-dependent manner. Hypotension as the main manifestation of circulatory collapse was proposed in about one-third of patients sustaining cardiac arrest during Holter monitoring and was more frequent at an advanced age.[11] The greater dangers of vasovagal syncope in older adults have also been emphasized by Alboni.[12] According to the authors' experience, frequent attacks, especially without prodrome in older age (>65 y), in patients with a history of reflex syncope at a young age should be considered a potential risk factor of vasovagal reflex-induced cardiac arrest.

Tilt testing is safe with rare complications.[1] However, ventricular fibrillation, acute myocardial ischemia, stroke, and transient cerebral ischemia have been reported.[13,14] Hypotension and bradycardia invoked by the vasovagal reflex may lead to profound cerebral hypoperfusion and alter electrophysiological properties of myocardial cells. Both may exert adverse effects on the functions of the heart and brain. This case suggests a possible role of vasovagal reflex in the pathogenesis of sudden cardiac arrest.

Another aspect of this case is that the first collapse on tilt was classified as VASIS 1 without severe bradycardia, but the second attack met VASIS 2B criteria, i.e., asystole >3 seconds, confirming the lack of reproducibility of the collapse pattern when using a laboratory challenge compared with recording spontaneous events by an implantable loop recorder, first shown by the International Study of Syncope of Uncertain Etiology.[15] Although our patient recovered from a VASIS Type 1 syncope when he was put in the supine position after losing consciousness, a VASIS Type 2B syncope attack soon followed, this was resolved by extracorporeal cardiac massage.

Conflicts of interest

All authors have no conflicts of interest to declare.

References

1. Moya A, Sutton R, Ammirati F, et al. Guidelines for the diagnosis and management of syncope (version 2009). The task force for the diagnosis and management of syncope of the European Society of Cardiology (ESC). *Eur Heart J*. 2009;30:2631–2671.
2. Rozenberg J, Wieling W, Schon IK, Westerhof B, Frampton C, Jardine D. MSNA during prolonged post-faint hypotension. *Clin Auton Res*. 2012;22:167–173.
3. Wieling W, Rozenberg J, Go-Schon IK, Karemaker JM, Westerhof BE, Jardine DL. Hemodynamic mechanisms underlying prolonged post-faint hypotension. *Clin Auton Res*. 2011;21:405–413.
4. Rea TD. Agonal respirations during cardiac arrest. *Curr Opin Crit Care*. 2005;11:188–191.
5. Wieling W, Rozenberg J, Schon IK, Karemaker JM, Westerhof BE, Jardine DL. Prolonged post-faint hypotension can be reversed by dynamic tension. *Clin Auton Res*. 2011;21:415–418.
6. Truhlář A, Deakin CD, Soar J, et al. European Resuscitation Council guidelines for resuscitation 2015: section 4. Cardiac arrest in special circumstances. *Resuscitation*. 2015;95:148–201.
7. Zysko D, Melander O, Fedorowski A. Vasovagal syncope related to emotional stress predicts coronary events in later life. *Pacing Clin Electrophysiol*. 2013;26:1000–1006.
8. Ruwald MH, Hansen ML, Lamberts M, et al. Prognosis among healthy individuals discharged with a primary diagnosis of syncope. *J Am Coll Cardiol*. 2013;61:325–332.
9. Ruwald MH, Numé AK, Lamberts M, et al. Incidence and influence of hospitalization for recurrent syncope and its effect on short- and long-term all-cause and cardiovascular mortality. *Am J Cardiol*. 2014;113:1744–1750.
10. Kruit MC, Thijs RD, Ferrari MD, Launer LJ, van Buchem MA, van Dijk JG. Syncope and orthostatic intolerance increase risk of brain lesions in migraineurs and controls. *Neurology*. 2013;80:1958–1965.
11. Watanabe E, Tanabe T, Osaka M, et al. Sudden cardiac arrest recorded during Holter monitoring: prevalence, antecedent electrical events, and outcomes. *Heart Rhythm*. 2014;11:1418–1425.
12. Alboni P. Therapy of vasovagal syncope: to prevent the vasovagal reflex or to treat the impending reflex? *Europace*. 2010;12:776–778.
13. Lepiece C, Dormal F, Blommaert D. Vasovagal syncope initiating ventricular fibrillation in a healthy subject. *Acta Cardiol*. 2013;68:635–638.
14. Matsushita K, Ishikawa T, Sumita S, et al. [Neurally mediated syncope with significant cardiac arrest and asymptomatic multiple cerebral infarctions: a case report]. *J Cardiol*. 2005;45:219–224.
15. Moya A, Brignole M, Menozzi C, et al. Mechanism of syncope in patients with isolated syncope and in patients with tilt-positive syncope. *Circulation*. 2001;104:1261–1267.